

THE CARDIAC IMPULSE AND THE MOTION OF THE HEART

BY

A. A. DELIYANNIS, P. M. S. GILLAM, J. P. D. MOUNSEY, AND R. E. STEINER

From the Departments of Medicine and of Radiology, and the Medical Research Council Cardiovascular Group, the Postgraduate Medical School of London

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"Quod erigitur cor, et in mucronem se sursem elevat, sic ut illo tempore ferire pectus, et foris sentiri pulsatio possit."

"The heart erects, and raises itself into a point, so that at this moment it strikes the chest wall, and externally a pulsation can be felt."

William Harvey, *Exercitatio anatomica de Motu Cordis et Sanguinis in Animalibus*. Frankfurt, 1628.

The apex beat and the character of the apical impulse are today universally accepted as basic and important physical signs: yet their mode of production remains only partially understood. Uncertainty exists as to which ventricle forms the apical impulse in health, Haycraft (1891) and Rushmer (1961) suggesting that it is formed by the right rather than the left ventricle, as is more usually held. The position of the apex beat is emphasized as of importance in the clinical assessment of heart size, yet the inconstant association between this point and the anatomical apex of the heart is less well known (Roesler, 1937). Finally, although the heaving sensation imparted to the hand by the hypertrophied left ventricle is familiar to all, the exact mechanism by which contraction of the hypertrophied heart produces this sensation is incompletely understood.

In this paper we have attempted to answer some of these questions. We have studied the genesis of the apical impulse in health and left ventricular hypertrophy, by recording the form of the impulse and relating this to the movements of the left ventricular cavity in timed left ventricular angiocardio-grams. In addition, using the dissection technique of Mall (1911) modified by Lev and Simkins (1956) to separate the heart muscle into different functional layers, we have shown how disturbance of the normal balance of forces of constriction and retraction within the separate myocardial functional layers may be responsible for the abnormal form of ventricular contraction in left ventricular hypertrophy.

THE INVESTIGATION

The *apical impulse* was recorded by the method described by Beilin and Mounsey (1962). The recording instrument, which is fixed to a rigid stand, contains a light metal rod, supported on light springs, which is applied to the chest wall in the axis of the movement to be recorded (Fig. 1). Displacement of the rod in its long axis varies the area of the photoelectric cell exposed to the light source. The current passed through the photoelectric cell is fed into a low frequency galvanometer and thus an optical record is obtained. The impulse recorder measures displacement of the site on the chest wall examined in relation to a fixed point in space (Dressler, 1937; Eddleman *et al.*, 1953; Mounsey, 1957). It does not measure relative displacement of a site in an intercostal space in relation to the surrounding chest wall, as in the conventional apex cardiogram (Marey, 1878; Mackenzie, 1902; Volhard, 1904; Weitz, 1917; Johnston and Overy, 1951; Luisada and Magri, 1952; Benchimol, Dimond, and Carson, 1961). To distinguish our record from the apex cardiogram, we have termed ours the *impulse cardiogram*, our aim being to produce a graphic record of the cadence of impulses felt by the hand.

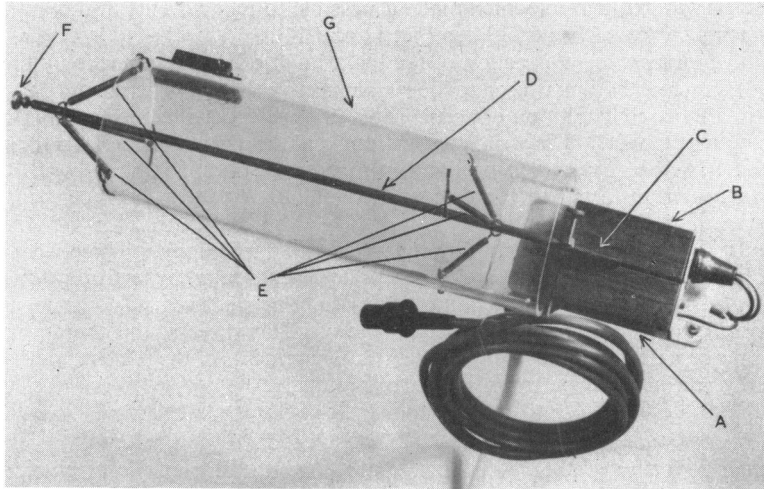


FIG. 1.—The impulse recorder. A=photoelectric cell. B=light source. C=metal flag, attached to metal rod, D. E=light springs suspending metal rod. F=knob applied to chest wall. G=perspex cylinder.

Details of the characteristics of the impulse recorder were given by Beilin and Mounsey (1962). A photoconductive cell is used that transmits sufficient current to require no amplification, being fed directly into the galvanometer. In routine recording the instrument is calibrated so that a displacement of 1 mm. at the chest wall produces a deflection of 3.5 cm. in the record. The instrument is so arranged that an upward deflection indicates outward movement of the chest wall. The frequency response of the recorder is satisfactory up to 10 cycles a second, giving a delay of up to 1/200th second, when tested on a sine-wave mechanical oscillator.

Impulse cardiograms were recorded with the patient propped up in bed at an angle of about 45° and also lying flat on a firm Dunlopillo mattress. The impulse was recorded from the site of greatest pulsation at the apex beat, determined by palpation and inspection. Impulse cardiograms were also routinely taken at the left parasternal area. A simultaneous phonocardiogram and electrocardiogram were recorded as reference tracings.

In all 230 impulse cardiograms were available for analysis. These included 60 patients with systemic hypertension, 40 with mitral valve disease, 20 with aortic valve disease, 40 with congenital heart disease, 10 with ischaemic heart disease, 10 with thyrotoxicosis, and 16 with cardiomyopathy. In addition 35 healthy control volunteers from among the medical staff were investigated.

Left ventricular angiocardiograms were taken on a bi-plane Elema roll film changer at six exposures a second. The exact time of each exposure was recorded on a simultaneous electrocardiogram. Selective injections were made either into the left ventricle by retrograde catheterization from the femoral artery, or into the left atrium by the transeptal route. In certain patients with much aortic incompetence, it was possible to visualize the left ventricular cavity after injection into the aorta. In 11 patients the site of the apex beat was recorded in the angiocardiogram, the marker being placed in position while the patient was lying flat on the angiocardiogram table. Studies were carried out on 26 patients, who were being investigated before possible cardiac surgery; impulse cardiograms and timed left ventricular angiocardiograms were available for analysis in each patient. The diagnosis was established by full clinical examination and investigation, the patients falling into three main groups: those with congenital heart disease, those with rheumatic heart disease, and lastly a group with cardiomyopathy. In the more detailed study of the motion of the left ventricle in relation to the form of the impulse, it was decided to include only 10 of the 26 patients, whose records were sufficiently free from extrasystoles following the injection of contrast medium to allow fair comparison of the angiocardiogram with the impulse cardiogram taken at a different time.

In the detailed study of the movements of the left ventricular cavity, superimposed tracings were made of angiocardiograms at three different moments in the cardiac cycle, at the end of diastole, in early systole, and at the end of systole. The exact timing of the three angiocardiograms chosen for analysis was determined

from the reference electrocardiogram, which made it possible to relate events in the impulse cardiogram to underlying movements of the left ventricle (see Fig. 11 and 14).

The Myocardial Architecture. Dissection of the heart muscle layers was made by the technique of Lev and Simkins (1956), in which boiling is used to loosen connective tissue. The uncut heart was simmered in a double saucepan in a 2 per cent solution of acetic acid. Simmering was continued for 3 to 7 hours, hypertrophied hearts requiring longer. The atria, pericardium, and the coronary arteries were next removed using forceps and swabs. The heart was then brought up through alcohol, beginning with 30 per cent for two days, after which it was transferred to 60 per cent alcohol. It remained in 60 per cent alcohol while the dissection was being carried out.

Twelve hearts from routine autopsies were prepared by this technique. Three were of normal weight, while the remaining nine showed varying degrees of left ventricular hypertrophy due to hypertension. The same routine of dissection was followed in every case. The right ventricle was separated from the left by blunt dissection in the posterior interventricular sulcus. This revealed the composite layered nature of the fibres forming the septal wall of the left ventricle. Measurements of the relative width of different muscle layers in the healthy and in the hypertrophied heart were made.

RESULTS

The Anatomical Relation of the Apex Beat to the Heart. Bi-plane left ventricular angiocardiograms, with the position of the apex beat recorded with a lead marker, were available for study in 14 patients: in 7, right ventricular angiocardiograms were also available; 5 of these had chronic rheumatic heart disease, 4 cardiomyopathy, and 4 congenital heart disease. In 1 patient with a basal systolic murmur the heart was found to be normal after full investigation.

TABLE I

No.	Sex and age	Diagnosis	Apex beat		Relation to cavity
			ICS	cm. to L.	Apex
1*	F 31	Functional systolic murmur	5	8	2 cm. above LV
2	M 49	Mitral incompetence and stenosis	5	10	5 " " "
3	F 42	Mitral stenosis and aortic incompetence	5	8.5	0 " " "
4*	F 57	Mitral incompetence	5	10	2 " " "
5	F 36	Aortic and mitral incompetence	5	10	0 " " "
6	M 53	Aortic incompetence	5	10	4 " " "
7*	M 26	Obstructive cardiomyopathy	5	12	0 " " "
8*	M 26	Obstructive cardiomyopathy	5	9.5	3 " " "
9	F 15	Obstructive cardiomyopathy	5	8.5	0 " " "
10	M 53	Obstructive cardiomyopathy	5	8	5 " " "
11	M 6	Ventricular septal defect	5	8	2 " " "
12*	M 29	Partial transposition	5	10	5 " " RV
13*	M 9	V.S.D. pulmonary hypertension	4	7	7 " " "
14*	M 12	V.S.D. pulmonary hypertension	6	10	2 " " "

*=RV as well as LV angiocardiograms. ICS=intercostal space.

Although the apex beat sometimes coincided with the cavity apex of the ventricle, in many patients it was found to be several centimetres above, on, or just inside, the lateral cardiac border in the postero-anterior view. Thus, while in 4 patients the position of the anatomical apex of the left ventricular cavity closely coincided with the height of the apex beat, in another 7 the apex beat lay between 2 and 5 cm. above the cavity apex (Table I) (Fig. 2 and 3). In the remaining 3 patients, in whom the enlarged right ventricle formed the apex beat, this lay between 2 and 7 cm. above the apex of the right ventricular cavity (Fig. 4). In our patients, therefore, *although the apex beat was related to the apical portion of the heart, it bore no constant relation to the apex of the ventricular cavity.*

The apex beat was seen to overlie the antero-septal ventricular wall in the normal angiocardiogram,

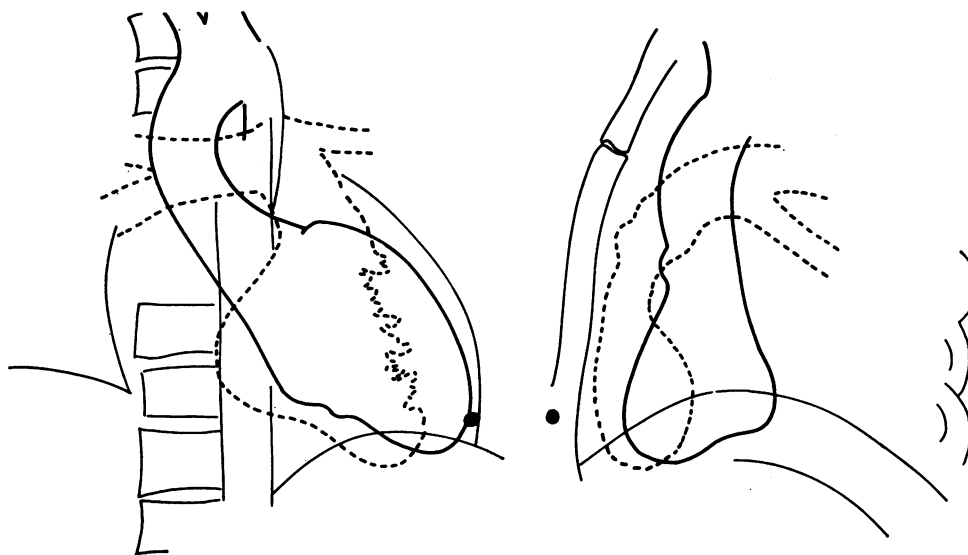


FIG. 2.—Superimposed tracings of right (dotted line) and left (solid line) ventricular angiocardigrams of normal subject with functional systolic murmur. The apex beat (black dot) lies 3 cm. anterior to the left ventricular cavity and 2 cm. lateral to the right ventricular cavity and overlies the antero-septal ventricular wall (antero-posterior and left lateral views).

being about the same distance anterior to the left ventricular cavity in the lateral view, as it was lateral to that of the right ventricle in the antero-posterior view (Fig. 2). In the normal heart, the apical impulse is thus formed primarily by left ventricular forces, since the septum consists predominantly of left ventricular muscle.

Much rotation of the interventricular septum may occur in right and left ventricular hypertrophy thus modifying the factors responsible for the apical and left parasternal impulses. In left ventricular hypertrophy, the septum may be rotated to the right, so that the antero-lateral wall of the left

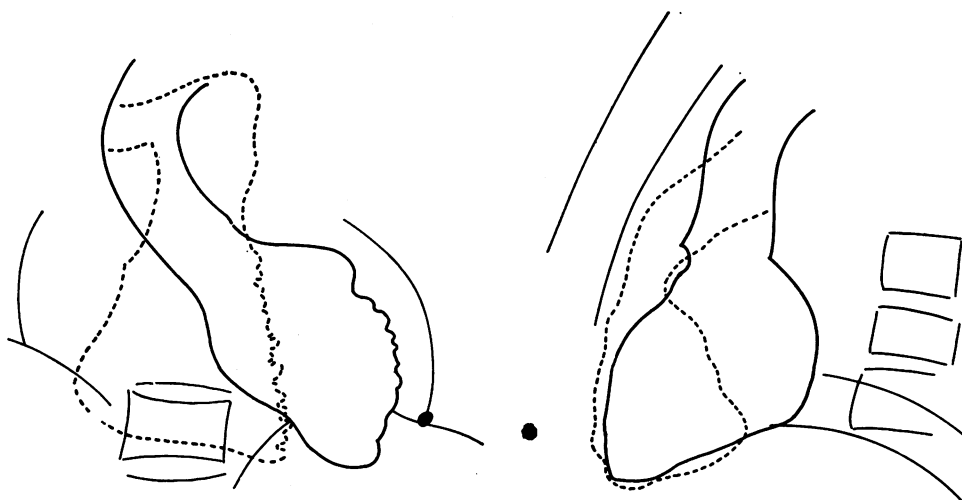


FIG. 3.—Superimposed tracings of right (dotted line) and left (solid line) ventricular angiocardigrams of patient with left ventricular hypertrophy from mitral incompetence. The apex beat (black dot) overlies the antero-lateral wall of the left ventricle and is 6 cm. lateral to the cavity of the right ventricle (antero-posterior and left lateral views).

ventricle underlies the apical impulse and the antero-septal wall underlies the parasternal impulse (Fig. 3). In such cases differentiation between right and left ventricular events by palpation of localized cardiac impulses becomes unreliable.

The converse of this condition is seen, when in great right ventricular hypertrophy and dilatation, the septum is rotated to the left. Here the right ventricle underlies not only the left parasternal impulse, but also the apical impulse (Fig. 4). Once again the differentiation between right and left ventricular hypertrophy on palpation becomes uncertain.

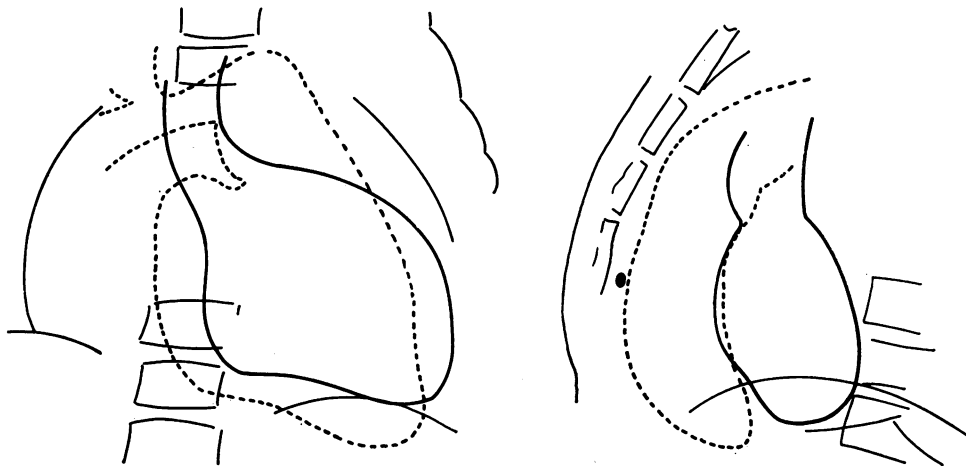


FIG. 4.—Superimposed tracings of right (dotted line) and left (solid line) ventricular angiocardiograms of patient with right ventricular hypertrophy from pulmonary hypertension and ventricular septal defect. The apex beat (black dot) in the 4th intercostal space, 7 cm. to the left of the mid-line, overlies the anterior wall of the right ventricle. The interventricular septum lies in the coronal plane, with the left ventricle rotated posteriorly (antero-posterior and left lateral views).

The Form of the Apical Impulse. The form of the apical impulse was found to be of three main types: the normal, the overacting, and the sustained.

(a) *The Apical Impulse in Health.* The characteristics of the apical impulse in health were described by Beilin and Mounsey (1962). They found that the apex beat lay in the fourth or fifth intercostal space not more than 10 cm. from the mid-line, that the excursion of the apical impulse was relatively small, and that its outward movement always terminated well before the second heart sound. The impulse cardiogram showed the length of the impulse in relation to the heart sounds and it was noted that it always ended before the last third of systole (Fig. 5).

In analysing the apical impulse, it was found useful to relate the deflection in the impulse record to the period of ventricular ejection. On this basis the impulse accompanying ventricular systole was divided into three portions. The pre-ejection period lay between the beginning of the Q wave in the electrocardiogram and the upstroke of the carotid pulse and included the periods of protosystole (Eddleman *et al.*, 1953) and isometric contraction. The ejection period covered the whole period of ventricular ejection. The post-ejection period included protodiastole and isometric relaxation (Wiggers, 1939). Two small outward movements were seen to compose the apical impulse. The first, the *pre-ejection beat*, lay in the pre-ejection period and began before, and finished after, the first heart sound. This was followed by a second outward movement, the *ejection beat*, whose upstroke just preceded the upstroke of the carotid pulse and which returned to the baseline well before the second heart sound. During the last third of systole and during the succeeding post-ejection period, there was often a small inward deflection, reflecting apical retraction. The impulse finally moved outwards again during the remainder of diastole.

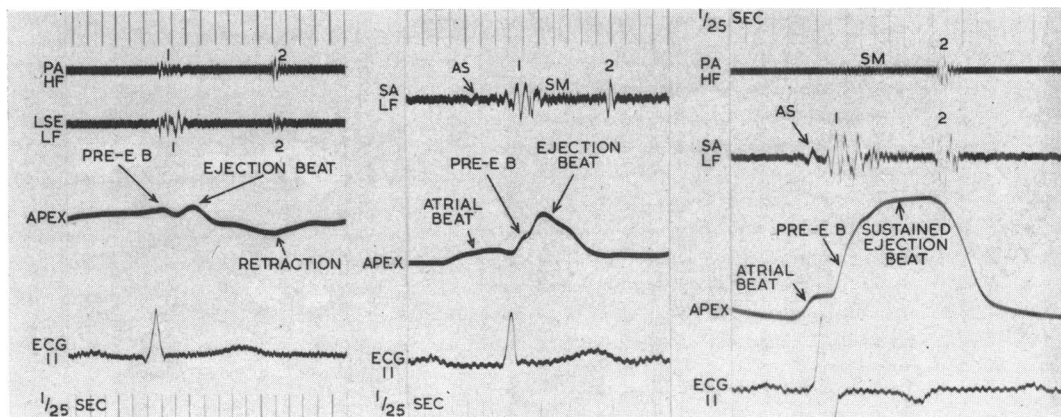


FIG. 5.—The apical impulse cardiogram (apex) in health. Pre-EB = pre-ejection beat. Phonocardiograms: PA, HF = pulmonary area, high frequency. LSE, LF = left sternal edge, low frequency. 1 and 2 = first and second heart sounds. Electrocardiogram lead II. The ejection beat returns to the baseline before the last third of systole, when slight systolic retraction is seen in this subject.

FIG. 6.—Apical impulse cardiogram (apex) of the overacting type, in thyrotoxicosis. Pre-EB = pre-ejection beat. Phonocardiogram: SA, LF = supramammary area, low frequency. AS = atrial sound. SM = systolic murmur. 1 and 2 = first and second heart sounds. Electrocardiogram lead II.

FIG. 7.—Apical impulse cardiogram (apex) of the sustained type, in hypertensive heart disease. Pre-EB = pre-ejection beat. Phonocardiograms: PA, HF = pulmonary area, high frequency. SA, LF = supramammary area, low frequency. AS = atrial sound. SM = systolic murmur. 1 and 2 = first and second heart sounds. Electrocardiogram lead II.

The amplitude of the impulse in the normal subject was not large, the greatest excursion in the impulse cardiogram being 1 cm. The influence of nutrition upon the amplitude of the apical impulse was not found to be important in the group of healthy subjects examined, there being no general relation between a tendency to be over- or underweight (Association of Life Insurance Directors and Actuarial Society of America, New York, 1912, quoted by Joslin *et al.*, 1959), and diminished or increased amplitude respectively of the apical impulse cardiogram.

(b) *The Overacting Impulse.* We defined an overacting impulse as one of increased amplitude but of normal form, and with a normal cadence of movements in impulse cardiogram (Fig. 6). Examples of the overacting impulse were met in health and disease. It was not uncommon in anxious children, whose thinner chest walls and narrower antero-posterior diameter of the thorax rendered the underlying cardiac movements more readily palpable. Adult subjects with a flat chest or depressed sternum also showed an overacting type of impulse. The diseases in which this overacting type of impulse was seen were those with an increased stroke output, as in thyrotoxicosis (Fig. 6), some cases of mitral incompetence, and some patients with a ventricular septal defect with a large left-to-right shunt (see Fig. 9).

(c) *The Sustained or Heaving Apical Impulse.* The sustained or heaving impulse of left ventricular hypertrophy was characterized in the impulse cardiogram by abnormal prolongation of the outward impulse up to, or beyond, the second heart sound (Fig. 7) (Beilin and Mounsey, 1962; Davie *et al.*, 1962). This prolongation is appreciated by the hand as a sustained quality and the degree of prolongation may be readily assessed clinically by relating the end of the outward impulse to the time of the second heart sound by simultaneous auscultation and palpation.

Increased amplitude of pulsation and displacement of the apex beat to the left were additional signs met only in the presence of the largest hearts and did not, therefore, constitute an essential component of the left ventricular type of impulse.

In the impulse cardiogram the pre-ejection and ejection beats were often fused into a single out-

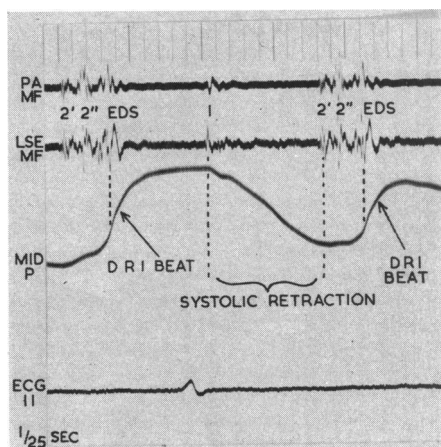


FIG. 8.—Præcordial impulse cardiogram (MID P) showing systolic retraction in constrictive pericarditis. The outward diastolic pulsation is the diastolic rapid inflow beat (DRI beat). Phonocardiogram: PA, MF=pulmonary area, medium frequency. 2' and 2''=split second heart sound. EDS=early diastolic sound.

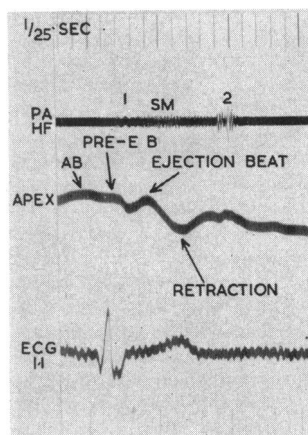


FIG. 9.—Apical impulse cardiogram (apex) of overacting type in ventricular septal defect (Patient A.W.). Significant systolic retraction following ejection beat. Atrial beat (AB) and pre-ejection beat (PRE-E B). Phonocardiogram (PA, HF) and electrocardiogram (lead II).

ward deflection, which was prolonged into the post-ejection period, the impulse only finally sinking to the base-line when isometric relaxation was complete (Fig. 7).

Prolongation of the apical impulse up to, or beyond, the second heart sound was a constant characteristic of all patients with a left ventricular heave of whatever cause, and was met in hypertension, aortic stenosis, aortic incompetence, and mitral incompetence. It was also sometimes seen in cardiomyopathy. Analysis of a series of 25 patients with hypertension, with and without left ventricular hypertrophy, showed that abnormal prolongation of the impulse corresponded closely with radiological and electrocardiographic evidence of hypertrophy, and is thus a useful clinical guide to the development of hypertrophy in hypertension (Beilin and Mounsey, 1962).

Another abnormal sign frequently met in association with a sustained apical impulse was in-

TABLE II

No.	Sex and age	Diagnosis	Hæmodynamic assessment	L.V. angio-cardiogram	Apical impulse
1	M 27	V.S.D.	(Small: reopened)	Retracts	Retraction
2	M 5	V.S.D.	4: 1 L-R shunt; PVR=2	Retracts	Retraction
3	F 20	V.S.D.	1:7: 1 L-R shunt; PVR=8	Retracts	Retraction
4	F 32	M.S. (M.I.)	PAP=57/17; PCP=30	Retracts	Sustained
5	F 38	M.S. (M.I.)	PAP=38/14; PCP=29	Retracts	Sustained
6	F 45	M.S. (M.I.)	PAP=32/20; PCP=22	Retracts	Sustained
7	M 49	M.I. (M.S.)	PAP=35/15; PCP=26	Protrudes	Sustained
8	F 32	M.I. (M.S.)	PAP=53/28; PCP=28	No retraction	Sustained
9	F 36	A.I. (A.S.) M.I. (M.S.)	PAP=77/32; PCP=24	No retraction	Sustained
10	M 53	A.I. (A.S.)	BP=112/50	No retraction	Sustained

PAP=pulmonary arterial pressure and PCP=wedged pulmonary capillary pressure in mm. Hg; PVR=pulmonary vascular resistance in units; V.S.D.=ventricular septal defect; M.S.=mitral stenosis; M.I.=mitral incompetence.

creased amplitude of the *atrial beat* accompanying atrial systole (Fig. 7) (Mackenzie, 1902; Harrison *et al.*, 1958; Mounsey, 1959; Parry and Mounsey, 1961; Beilin and Mounsey, 1962; Dimond and Benchimol, 1963). The atrial beat, although present in health, is usually too small to be felt. In left ventricular hypertrophy, however, it is often increased in amplitude and is felt as a sustained presystolic outward beat immediately preceding the major sustained impulse accompanying ventricular systole.

A sustained apical impulse is not pathognomonic of ventricular hypertrophy, since it is also met in some cases of cardiac infarction, where a paradoxical bulge of a weakened portion of the myocardium seems to be responsible (Harrison, 1959). It is also sometimes recorded in cardiac aneurysm, either at the apex or elsewhere over the præcordium. Differentiation from the præcordial diastolic beat of constrictive pericarditis (Potain, 1856; McKusick, 1952; Mounsey, 1957), sustained throughout diastole, can always be made by careful timing at the bed-side (Fig. 8).

The Motion of the Left Ventricle. The 10 patients, whose left ventricular angiocardiograms were selected for study, were divisible into three groups (Table II). Three patients had a ventricular septal defect; their impulse was of the overacting type, showing a large ejection beat, followed by a retraction in the latter part of systole. Four had chronic rheumatic heart disease, in 2 of whom there was calcific mitral valve disease with predominant incompetence, in 1 gross aortic and mitral incompetence, and in 1 great aortic incompetence alone. These 4 patients all had great left ventricular hypertrophy and a sustained apical impulse. Finally there was a group of 3 patients with mitral stenosis, whose apical sustained impulse was due to right ventricular hypertrophy, with enlargement of the right heart leftwards to form the apex, the left ventricle being displaced posteriorly.

Fundamental differences in the behaviour of the left ventricular cavity at the end of systole were seen in the patients with the overacting, as opposed to the sustained, impulse. While in the overacting group free backward retraction of the apical portion of the heart in late systole was shown, in



FIG. 10.—Left ventricular angiocardiograms (lateral view) of ventricular septal defect (Patient A.W.), in diastole (left) and systole (right). Apical systolic retraction demonstrated.

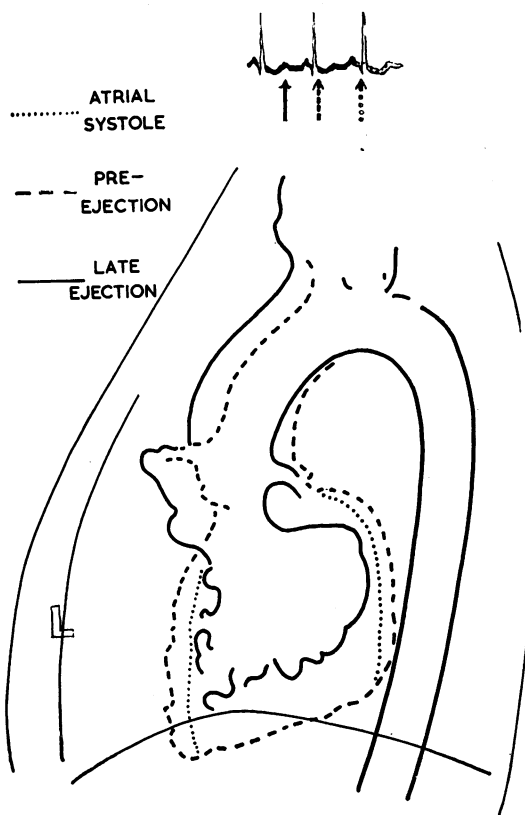


FIG. 11

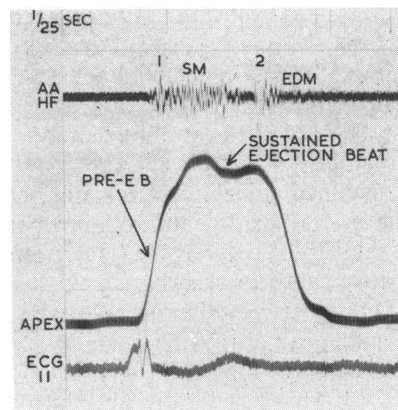


FIG. 12

FIG. 12.—Apical impulse cardiogram (apex) of sustained type in aortic and mitral incompetence (Patient I.F.). Sustained ejection beat of large amplitude. Pre-ejection beat (PRE-E B). Phonocardiogram: AA, HF=aortic area-high frequency. SM=systolic murmur. EDM=early diastolic murmur. Electrocardiogram lead II.

FIG. 11.—Superimposed tracings of lateral angiocardigrams of Patient A.W., at end of atrial systole (.....), during pre-ejection period (-----), and in late systole (——). Exact timing of exposures marked with arrows on simultaneous electrocardiogram. Forward movement of anterior wall of left ventricle in pre-ejection period. Concentric retraction of apex in late systole. L=position of apex beat.

those with a sustained impulse the apex failed to retract—and indeed in one it actually approached more closely to the anterior chest wall.

Detailed movements of the left ventricular cavity in relation to the impulse records are illustrated in selected angiocardigrams. The patient with the overacting impulse was a boy of 5 years with a 4:1 shunt through the ventricular septal defect and a low pulmonary vascular resistance of 2 units. His impulse record (Fig. 9) shows relatively large amplitude, with a steep ejection beat followed by retraction in the latter half of systole. His systolic and diastolic left ventricular angiocardigrams are illustrated in Fig. 10. The movement responsible for the apical impulse takes place nearly in an antero-posterior axis and so is best visualized in the lateral view, of which serial superimposed tracings have been made (Fig. 11). The exact timing of the pictures selected for tracing is shown by the arrows on the inset simultaneous electrocardiogram. The site of the apex beat on the anterior chest wall is shown by the capital letter L. Three positions of the cavity outline are drawn: the dotted line represents its position at the very end of diastole, coinciding with the beginning of the Q wave in the electrocardiogram; the interrupted line shows the cavity during the period of pre-ejection, 0.6 sec. after the Q wave. The forward movement of the anterior wall during the periods of pre-ejection and early ejection was a constant finding in all patients. It is this forward movement of the anterior wall of the left ventricle that is responsible for the outward impulse that we call the apex beat. Finally, the solid line indicates the position of the cavity in late systole at the time of the T wave in the electrocardiogram. Concentric contraction of the whole of the ventricular cavity has taken place and in particular the apex has retracted from the anterior chest wall—thus causing, in turn, retraction of the apical impulse.

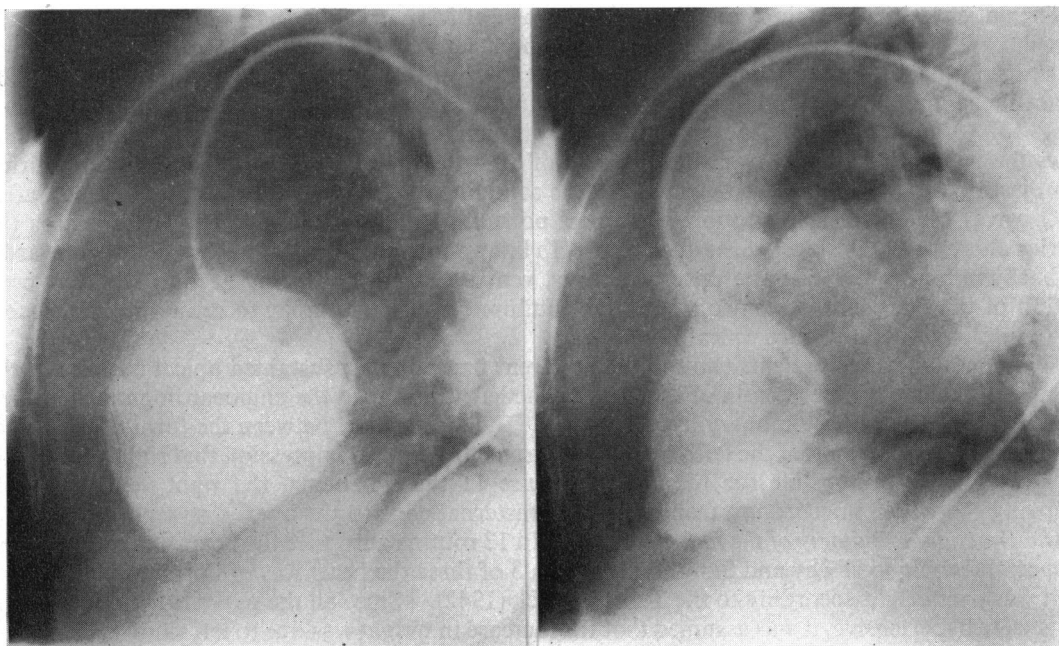


FIG. 13.—Left ventricular angiocardiograms (lateral view) of aortic and mitral incompetence (Patient I.F.), in diastole (left) and systole (right). Failure of backward systolic retraction of apex (left ventricular injection).

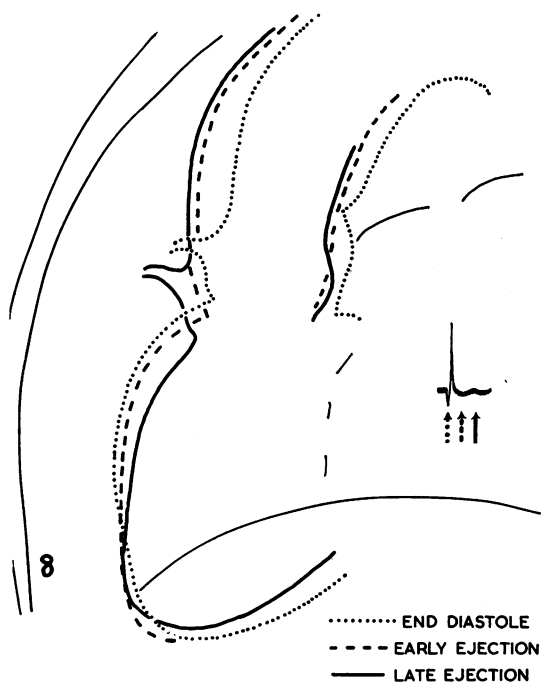


FIG. 14

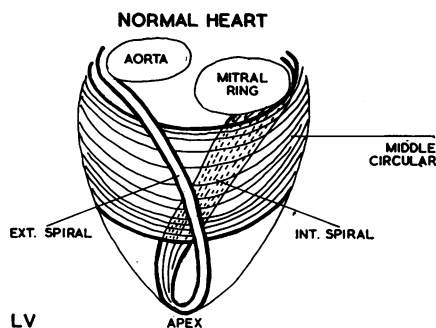


FIG. 15

FIG. 15.—Diagrammatic representation of layers of fasciculi composing left ventricular wall. The external and internal spiral fasciculi (single diagrammatic strand shown) are continuous through whorl of fibres at apex. The middle fasciculus lies between the two.

FIG. 14.—Superimposed tracings of lateral angiocardiograms of Patient I.F. at the end of diastole (.....), during early ejection (-----), and in late ejection (——). Exact timing of exposures marked with arrow on simultaneous electrocardiogram. Forward movement of anterior wall of left ventricle in early ejection period. Failure of backward retraction of apex in late ejection period. Opacification of left atrium from mitral incompetence. 8=position of apex beat (aortic injection).

The patient with a sustained impulse was a woman of 36 years, suffering from chronic rheumatic heart disease, with dominant aortic and mitral incompetence. Her impulse record (Fig. 12) shows a sustained apical impulse of large amplitude that does not return to the baseline until after the time of the second heart sound. Systolic and diastolic left ventricular angiocardiograms are shown in Fig. 13. No retraction of the apex of the cavity is seen in late systole. In Fig. 14, the dotted line shows the position of the cavity wall at the end of diastole, the interrupted line its position during early ejection (0.18 sec. after the Q wave) and the solid line that at the end of systole (0.36 sec. after the Q wave). During early ejection, the apical portion of the cavity moves forward towards the anterior chest wall at the level of the apex beat. In late systole, the apex withdraws slightly upwards, but backward systolic retraction does not occur, ventricular ejection being accomplished by contraction of the body and descent of the base. Failure of the cavity apex to retract in late systole results in a sustained outward apical impulse.

The remaining three patients showed the apparent paradox of a sustained apical impulse in the presence of late systolic retraction of the apex of the left ventricle in the angiocardiogram: all three had mitral stenosis with pulmonary hypertension. The discrepancy between the form of the apical impulse and the movement of the left ventricle confirmed the clinical impression that right ventricular hypertrophy was responsible for the apical impulse in these patients, the right ventricular lift occupying the whole præcordium from the left parasternal edge to the apex.

The Functional Anatomy of the Left Ventricle. In 12 routine autopsies the heart was examined by the special technique of Lev and Simkins (1956): in 3 of these the heart was of normal weight, while in 9 it was overweight according to the Table of Zeek (1942). Since all the patients with overweight hearts were hypertensive, it was assumed that the increase in weight was due to left ventricular hypertrophy. According to Lev and Simkins (1956) the myocardial architecture is made up of three main layers, which they call the epicardial, the middle, and the endocardial fasciculi. These three layers are distinguished by the different direction of the fibres in each, but the dividing line between each is indistinct. In order to emphasize this basic difference in direction of fibres, we have preferred to call them the external spiral, the middle circular, and the internal spiral fasciculi (Fig. 15). In the belief that hypertrophy of the middle circular layer might contribute to the altered manner of contraction in left ventricular hypertrophy, we made measurements of the width of the middle circular fasciculus in the interventricular septum, relating this to the over-all length of the heart from apex to base.

In Fig. 16A is seen the appearance of the normal heart when dissected through the posterior interventricular groove. Left and right ventricle have been partially separated, the left ventricle lying on the left of the picture. At its upper basal border, the cut edge of the external spiral fasciculus is seen, which is coursing round the anterior surface of the left ventricle to form a whorl at the apex. Its fibres are then continued as the internal spiral fasciculus which can be seen at the apex of the left ventricle. These fibres tuck in underneath the middle circular fasciculus, which wraps round the upper three-fifths of the left ventricle. In Fig. 16B the fibres of the middle circular fasciculus have been cut and reflected to reveal the internal spiral fasciculus, whose fibres are coursing diagonally upwards.

It will be seen, therefore, that while the base and equator of the heart are amply supplied with circular fibres of the middle circular fasciculus, the apex is largely composed of a continuous whorl of spiral fasciculi, whose internal and external fibres cross each other at right angles. Sparing of the apical two-fifths of the heart by the middle circular fasciculus was a constant finding in hearts of normal weight (Table III). In the more hypertrophied hearts, increasing width of the middle circular band of muscle was found in relation to the total length of the heart (Table III). As a result the middle circular fasciculus encroached more closely upon the apex and in the heaviest heart with marked left ventricular hypertrophy it appeared to extend right down to the apex (Fig. 17).

The likely consequences of increasing width of the middle circular fasciculus in left ventricular hypertrophy on the functional anatomy of the left ventricle are illustrated in Fig. 18. The

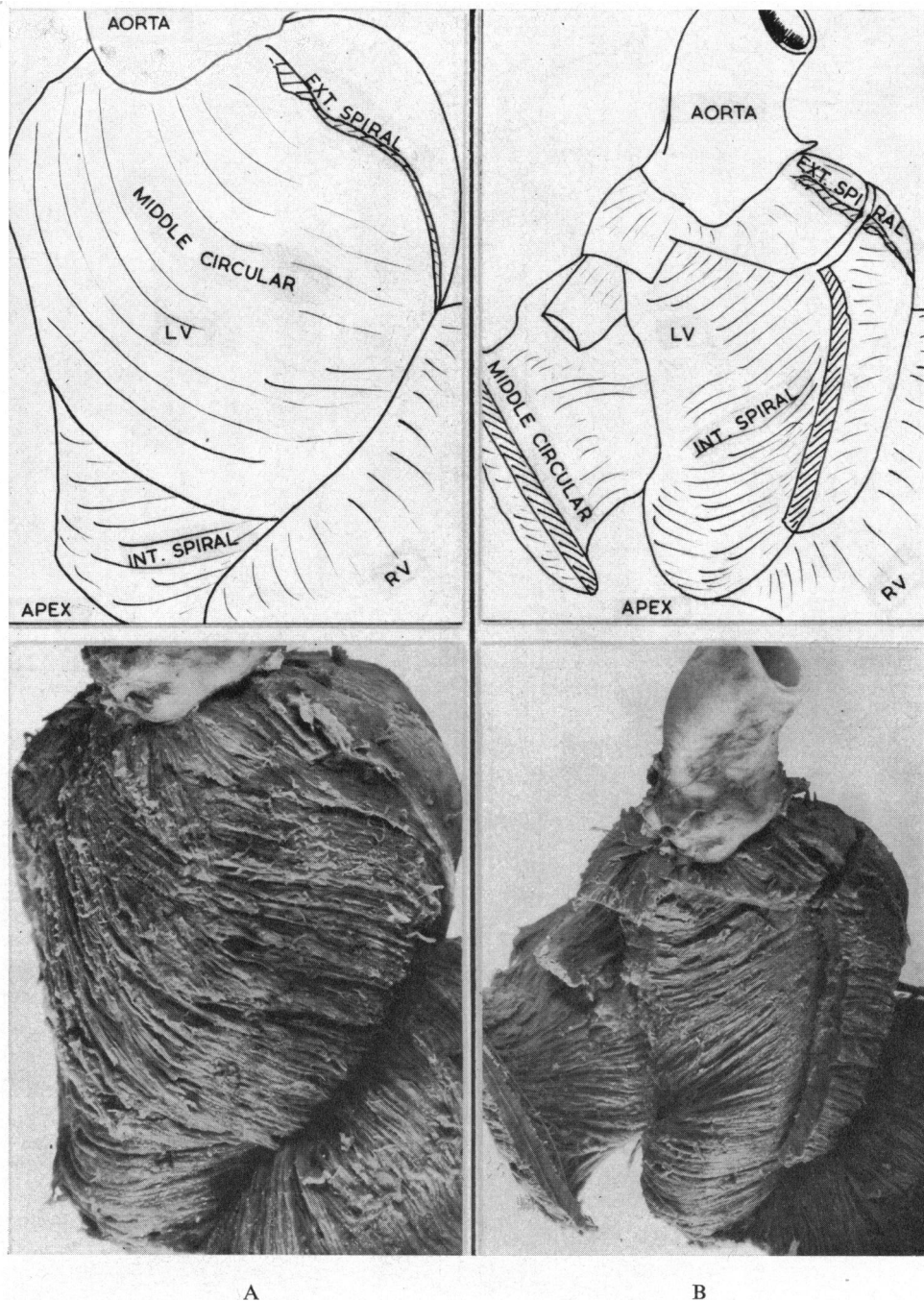


FIG. 16.—Dissection of normal heart. Posterior view. Left and right ventricles peeled apart through posterior inter-ventricular septum. *Left-hand column* (photograph below, tracing above); before division of middle circular fasciculus of left ventricle. *Right-hand column*; after division and reflection of middle circular fasciculus, to reveal upward-coursing fibres of underlying internal spiral fasciculus. Note middle circular fasciculus does not extend to cardiac apex.

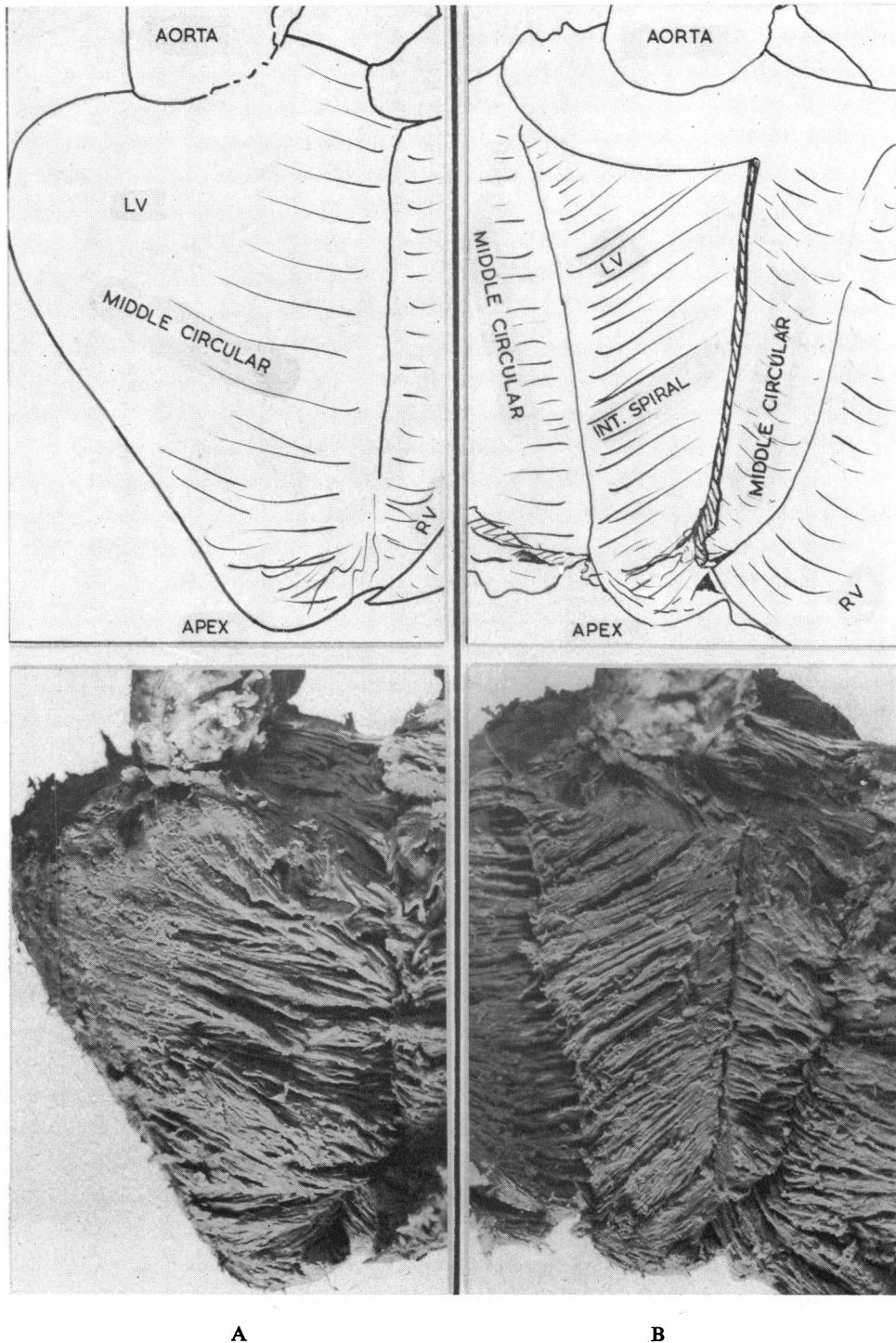


FIG. 17.—Dissection of heart with left ventricular hypertrophy from malignant hypertension. Posterior view. Left and right ventricles peeled apart through posterior interventricular septum. *Left hand column* (photograph below, tracing above); before division of middle circular fasciculus of left ventricle. *Right-hand column*; after division and reflection of middle circular fasciculus. Note middle circular fasciculus extends to cardiac apex.

TABLE III

No.	Sex and age		Diagnosis	Blood pressure (mm. Hg)	Heart weight (g.)	Heart length* (cm.)	Width of middle circular fasciculus (cm.)
1	M	56	Reticulum cell sarcoma	Normal	315(317)*	5.3	3.0
2	M	56	Pulmonary tuberculosis	Normal	320(317)	5	3.2
3	M	46	Carcinoma prostate	Normal	335(327)	5.5	3.0
4	F	64	Carcinoma bronchus	160/90	350(268)	5	3.5
5	F	77	Carcinoma breast	270/160	359(254)	5.3	3.2
6	M	84	Diabetes	150/100	422(340)	5	4.0
7	F	69	Carcinoma larynx	150/100	435(247)	6.3	3.7
8	M	59	Post-op. pulm. embolus	185/100	439	7	7.0
9	F	50	Hypertension	230/125	450(254)	7	6.4
10	M	37	Cholangitis	Terminal	455(336)	7	5.0
11	M	57	Dissecting aneurysm	310/110	560(317)	7.5	7.5
12	M	29	Malignant hypertension		590(340)	8	8.0

Figures in brackets are normal heart weights for height and sex = ± 30 (Zeek, 1942).

* Measurements of heart length and width of middle circular fasciculus made after shrinkage from boiling.

arrangement of the muscle fasciculi in health are such that the constricting action of the circular fibres would act predominantly upon the upper three-fifths of the ventricle, while retraction of the apex would be accomplished by the spiral whorl at the apex. In the presence of hypertrophy, however, the increased bulk of the middle circular fasciculus extending down to the apex would inhibit the normally unopposed retracting action of the spiral apical fibres. Indeed the constricting action of the widened middle circular fasciculus would cause the whole heart to erect, with the result that the apex might actually protrude rather than retract.

Although extension of the middle circular fasciculus toward the apex may well be one important

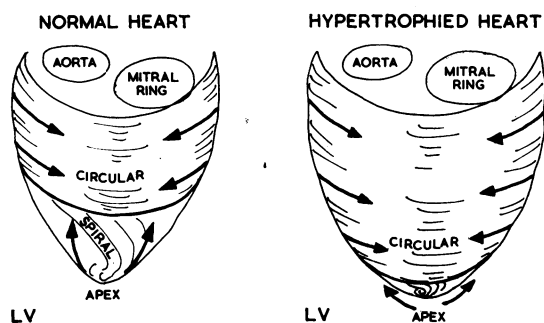


FIG. 18.—Schema of forces resulting from contraction of middle circular and of spiral fasciculi of left ventricle. In health, the middle circular fasciculus constricts the upper portion of the heart, while the relatively unopposed spiral fibres retract the apex. In left ventricular hypertrophy, the widened middle circular fasciculus extends further toward the apex, thus tending to inhibit the apical retracting action of the spiral fasciculus.

FIG. 19.—Drawing of left hemithorax (after Gray's Anatomy, 1949).

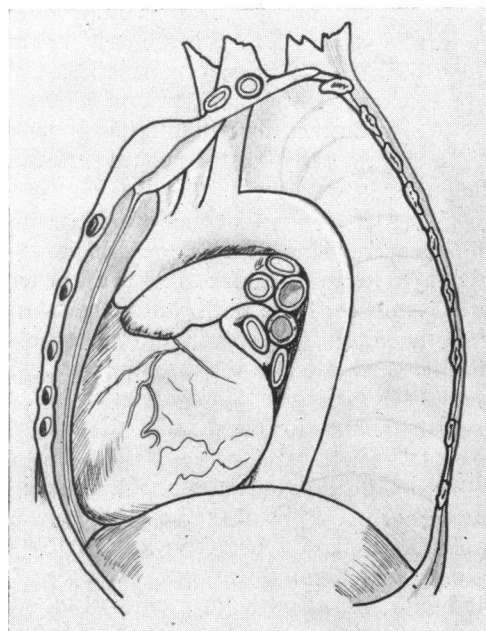


FIG. 19.

factor in failure of retraction of the cardiac apex in left ventricular hypertrophy, it is probable that ventricular dilatation with incomplete ventricular emptying also plays a part in some patients. All four patients in our series with a sustained apical impulse and failure of apical retraction in the angiogram were also noted to have some dilatation of the left ventricle (Table II and Fig. 13).

The heart lies upon the anterior shelf of the diaphragm, with the apex and anterior wall of the heart held in close proximity to the anterior chest wall (Fig. 19). It can be seen how forward movement of the apex and antero-septal heart wall, in the pre-ejection and early ejection periods, tends to press the heart against the anterior chest wall, thus giving rise to the normal short apical impulse. In left ventricular hypertrophy failure of retraction or even protrusion of the apical portion of the heart in late systole causes continued pressure of the heart against the anterior chest wall throughout systole, with the result that the examining hand on the præcordium feels a sustained heaving apical impulse.

SUMMARY AND CONCLUSIONS

The genesis of the apical impulse in health and in left ventricular hypertrophy has been studied, using the impulse recorder described by Beilin and Mounsey (1962) and serial timed left ventricular angiograms. In addition a detailed study of the myocardial architecture in normal and hypertrophied hearts was made, using the technique of layered muscle dissection of Lev and Simkins (1956).

The apex beat, marked in bi-plane left ventricular angiograms was found to approximate to the apex and antero-septal wall of the left ventricle. It bore no constant relation to the apex proper of the ventricular cavity, being often several centimetres above it.

As a result of marked rotation of the septum in ventricular hypertrophy, the right or the left ventricle sometimes enlarged so as to encroach on the position normally occupied by the other ventricle. Thus the apex beat could be formed by the right ventricle in right ventricular hypertrophy: conversely the left parasternal impulse could be formed by the left ventricle in left ventricular hypertrophy.

There were three main forms of the apical impulse: the normal, the overacting, and the sustained. In health the normal impulse consisted of outward movement followed by retraction in the last third of systole. In the overacting heart, the impulse showed increased amplitude; this was seen both in thin, anxious, healthy subjects, and in the overacting heart of thyrotoxicosis, and in conditions with a left-to-right intracardiac shunt. Thirdly, a sustained outward impulse, extending throughout systole and even into early diastole, characterized left ventricular hypertrophy in hypertension and chronic rheumatic heart disease.

In the left ventricular angiogram, the movements of the heart responsible for the cardiac impulse were visualized. In ventricular septal defect with an overacting impulse, the anterior wall of the left ventricle moved forward in early systole: hence the initial outward apical impulse. In later systole the heart both contracted in diameter and retracted in length, so that the apex withdrew from the anterior chest wall and the impulse moved inwards. In left ventricular hypertrophy from chronic valve disease, the essential abnormality responsible for the sustained apical impulse in late systole was seen to be failure of backward retraction of the cardiac apex, which thus remained pressed against the anterior chest wall. Incomplete left ventricular emptying was also noted in the latter patients.

Layered dissection of the healthy heart showed that the middle circular fasciculus covered only the basal three-fifths of the heart, the lower two-fifths and the apex being composed almost entirely of spiral fasciculi. In left ventricular hypertrophy the middle circular fasciculus was found to be broader, extending down toward the apex. Its increased width would thus tend to inhibit the normally unopposed retracting action of the spiral fasciculi at the apex, and cause the apex to protrude, thus suggesting one cause for failure of apical retraction in the angiogram and for the heaving, sustained apical impulse of left ventricular hypertrophy.

We would like to thank Professor Goodwin for allowing us to include many of his patients in our study, Professor Harrison and the staff of the Department of Morbid Anatomy for their help with the anatomical dissections, and Dr. Shillingford and the staff of the M.R.C. Cardiovascular Unit, who designed and built the impulse recorder. Finally we wish to thank Miss Jean Powell for technical, and Miss Joan Rooker for secretarial, assistance.

REFERENCES

- Association of Life Insurance Directors and Actuarial Society of America, New York (1912), pp. 38 and 67. Published by a committee. Quoted by Joslin, E. P., Root, H. F., White, P., and Marble, A. (1959). *The Treatment of Diabetes Mellitus*, 10th ed. Kimpton, London.
- Beilin, L., and Mounsey, P. (1962). The left ventricular impulse in hypertensive heart disease. *Brit. Heart J.*, **24**, 409.
- Benchimol, A., Dimond, E. Grey, and Carson, J. C. (1961). The value of the apexcardiogram as a reference tracing in phonocardiography. *Amer. Heart J.*, **61**, 485.
- Davie, J. C., Langley, J. O., Dodson, W. H., and Eddleman, E. E. (1962). Clinical and kinetocardiographic studies of paradoxical precordial motion. *Amer. Heart J.*, **63**, 775.
- Dimond, E. Grey, and Benchimol, A. (1963). Correlation of intracardiac pressure and præcordial movement in ischaemic heart disease. *Brit. Heart J.*, **25**, 389.
- Dressler, W. (1937). Pulsations of the wall of the chest. *Arch. intern. Med.*, **60**, 225, 437, 441, 654, and 663.
- Eddleman, E. E., Willis, K., Reeves, T. J., and Harrison, T. R. (1953). The kinetocardiogram I. Method of recording præcordial movements. *Circulation*, **8**, 269.
- Gray's Anatomy (1949). 30th ed., ed. T. B. Johnston, and J. Whillis, p. 772. Longmans Green, London, New York, Toronto.
- Harrison, T. R. (1959). Some clinical and physiologic aspects of angina pectoris. *Johns Hopk. Hosp. Bull.*, **104**, 275.
- , Lowder, J. A., Hefner, L. L., and Harrison, D. C. (1958). Movements and forces of the human heart. *Circulation*, **18**, 82.
- Harvey, W. (1628). *Exercitatio anatomica de Motu Cordis et Sanguinis in Animalibus*. Frankfurt.
- Haycraft, J. B. (1891). The movements of the heart within the chest cavity and the cardiogram. *J. Physiol. (Lond.)*, **12**, 438.
- Johnston, F. D., and Overy, D. C. (1951). Vibrations of low frequency over the precordium. *Circulation*, **3**, 579.
- Lev, M., and Simkins, C. S. (1956). Architecture of the human ventricular myocardium: technic for study using a modification of the Mall-MacCallum method. *Lab. Invest.*, **5**, 396.
- Luisada, A. A., and Magri, G. (1952). The low frequency tracings of the precordium and epigastrium in normal subjects and cardiac patients. *Amer. Heart J.*, **44**, 545.
- Mackenzie, J. (1902). *The Study of the Pulse, Arterial, Venous and Hepatic, and of the Movements of the Heart*. Pentland, Edinburgh and London.
- McKusick, V. A. (1952). Chronic constrictive pericarditis. II. Electrocardiographic studies and correlations with roentgenkymography, phonocardiography, and right ventricular pressure curves. *Johns Hopk. Hosp. Bull.*, **90**, 27.
- Mall, F. P. (1911). On the muscular architecture of the ventricles of the human heart. *Amer. J. Anat.*, **11**, 211.
- Marey, E. J. (1878). *La Méthode graphique dans les Sciences expérimentales*. Masson, Paris.
- Mounsey, P. (1957). Præcordial ballistocardiography. *Brit. Heart J.*, **19**, 259.
- (1959). Præcordial pulsations in relation to cardiac movement and sounds. *Brit. Heart J.*, **21**, 457.
- Parry, E., and Mounsey, P. (1961). Gallop sounds in hypertension and myocardial ischaemia modified by respiration and other manœuvres. *Brit. Heart J.*, **23**, 393.
- Potain, C. (1856). Adhérence du péricarde: triple bruit du cœur: impulsion diastolique. *Bull. Soc. anat. Paris*, **31** (2 ser., 1), 378.
- Roesler, H. (1937). *Clinical Roentgenology of the Cardiovascular System*. Baillière, Tindall and Cox, London.
- Rushmer, R. F. (1961). *Cardiovascular Dynamics*, 2nd ed. Saunders, Philadelphia and London.
- Volhard, F. (1904). Ueber Leberpulse und über die Compensation der Klappenfehler. *Berl. klin. Wschr.*, **41**, 522.
- Weitz, W. (1917). Über die Kardiographie am gesunden Herzen mit dem Frank'schen Apparat. *Dtsch. Arch. klin. Med.*, **124**, 134.
- Wiggers, C. J. (1939). *Physiology in Health and Disease*, 3rd ed. Kimpton, London.
- Zeek, P. M. (1942). Heart weight. *Arch. Path.*, **34**, 820.